Physiology & Behavior 141 (2015) 199–206

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

Sleep and the endogenous melatonin rhythm of high arctic residents during the summer and winter

Michel A. Paul [a], Ryan J. Love [a,*], Andrea Hawton [a], Josephine Arendt [b]

[a] Defence Research and Development Canada, Toronto, ON, M3K2C9, Canada
[b] University of Surrey, Guildford, Surrey GU2 7XH, United Kingdom

HIGHLIGHTS

- Arctic residence has been associated with poor sleep in summer and winter.
- Summer and winter trials were conducted to evaluate seasonal effects on sleep.
- Several subjects in June had a delayed melatonin rhythm and sleep difficulty.
- Excessive evening light exposure likely caused the circadian phase delay observed.

ARTICLE INFO

Article history:
Received 21 October 2014
Received in revised form 15 January 2015
Accepted 20 January 2015
Available online 20 January 2015

Keywords:
Polar regions
Dim light melatonin onset
Circadian rhythms
Fatigue

ABSTRACT

The seasonal extremes of photoperiod in high latitudes place particular strain on the human circadian system. Arctic residence has been associated with poor sleep in both summer and winter. The goal of the work reported here was to study the circadian rhythms of individuals living in the high Arctic by measuring sleep variables and the timing of melatonin production. Two research trials were conducted in the built environment of CFS Alert (82° 29′ 58″ N). Participants wore motion logging devices (actigraphs), which measure ambient light as well as motion, for 1 week to provide data on sleep quantity, quality and light exposure. On the penultimate day of each trial, the participants were maintained together in a gymnasium with lounge chairs and saliva was collected at regular intervals to measure melatonin and assess the dim light melatonin onset (DLMO), offset (MelOFF), 50% rise and fall times of the whole profile and total production. In general, sleep duration was found to be significantly different between the January and June data collections at CFS Alert, with participants in June sleeping 50 min on average less each day compared to their January counterparts. In June sleep was mistimed in many subjects relative to circadian phase as evidenced by the melatonin rhythm. Exposure to bright evening light was the most likely causal factor and should be avoided in the Arctic summer. The Arctic summer represents a particularly challenging environment for obtaining sufficient sleep. This has implications for the cognitive performance of staff during work hours.

1. Introduction

The high Arctic is a particularly strenuous environment for the human circadian system due to the lack of light in the winter months and lack of darkness in the summer months. Of the many problems that may arise from such extremes in photoperiod, greater negative affectiveness and fatigue are common in the winter months and mild insomnia is common in both the summer and winter months [1].

The human circadian system is regulated by external time cues or zeitgebers, the major influence being the light–dark cycle. The production of melatonin is the best marker for circadian timing. Suppression of endogenous melatonin production caused by nighttime exposure to bright light, and/or desynchrony of endogenous melatonin production with an individual's rest–work cycle, may lead to mild/moderate insomnia [2]. There is also evidence to indicate that the quantity and duration of melatonin production are greater during the winter months at high latitudes [3–6], and that increased duration of melatonin production contributes to fatigue and increased negative affect [7].

We have recently completed a 4-year project to evaluate, and provide a framework for, the manipulation of the circadian system forwards or backwards to minimize fatigue among Canadian Forces personnel during critical military operations [8–10]. This capability can be applied to help those military members who are posted in the high Arctic during the seasonal extremes of photoperiod (24 h darkness or 24 h sunlight) by providing the necessary circadian system time cues that are otherwise absent. The purpose of the work reported here was to

* Corresponding author at: 1133 Sheppard Ave. West, Defence Research and Development Canada, Toronto, ON M3K2C9, Canada.
E-mail address: ryan.love@drdc-rddc.gc.ca (R.J. Love).

http://dx.doi.org/10.1016/j.physbeh.2015.01.021
0031-9384/

document endogenous melatonin production in Canadian Forces personnel deployed to the high Arctic to develop a baseline understanding of the circadian system in these circumstances.

2. Methods

2.1. Subject inclusion/exclusion criteria/age/gender demographics

This report covers two high Arctic trials as follows:

1) January 14th to 22nd, 2012, Canadian Forces Station (CFS) Alert (82° 29′ 58″ N)
2) June 8th to 17th, 2012, CFS Alert.

To qualify, the subjects had to have been at CFS Alert for at least 1 month. The subjects provided written informed consent prior to their participation in the protocol, which was approved by the DRDC Human Research Ethics Committee and met the ethical standards of the Declaration of Helsinki. Subjects were compensated for their participation in accordance with Canadian Government Guidelines for experimental stress allowance.

The January trial included 14 subjects, 9 men and 5 women, with an age range from 21 to 56 years. The mean age and standard deviation of the participants was 36.9 ± 2.9 years. The June trial included 12 different subjects, 8 men and 4 women, with an age range from 22 to 47 years. The mean age and standard deviation of the participants was 31.2 ± 2.8 years. Data from the June trial was previously reported in [11].

2.2. Data collection protocol

The subjects wore actigraphs (Motionlogger, version 14.000, Ambulatory Monitoring Inc., Amherst, NY, USA) for the entire duration of the trial, as listed above for January and June trials. The actigraphs were used to record various measures of sleep including time of sleep onset, total duration of sleep, sleep efficiency, arise time, total wake minutes after sleep onset, and the number of sleep episodes during the main sleep period. Such use of actigraphy for the study of circadian rhythms and sleep has been well established [12]. The subjects also maintained a sleep log where they recorded caffeine and alcohol consumption, naps, and subjective estimates of sleep onset and wake times for the entire duration of the trial. On the penultimate day of the trials (Saturday January 21st or Saturday June 16th), the subjects arrived at the Station gymnasium for 0830 h. They were assigned lounge chairs for the 24-h saliva collection period. Gymnasium lights were turned off but supplementary lighting (to 5 lx in the angle of gaze) was stationed around the gymnasium and in the washrooms. A large monitor was set up about 20 ft in front of the lounge chairs to provide the subjects with movies during the 24-h period. Eye-level ambient light from the monitor was less than 5 lx for all subjects while at their chairs, and they were not permitted to get close enough to the monitor to exceed this light threshold. The subjects were allowed to get up from their chairs to go to the washrooms or socialize for about 100 min of every 2 h, while remaining in areas with ambient light of less than 5 lx. They had to be back in semi-recumbent posture in their lounge chairs for the 15 min prior to each sample and no eating or drinking was permitted during this time. Snacks were available to the subjects throughout the data collection and meals were provided at 1200 h and 1700 h. The subjects were not permitted to consume any caffeine-containing substances, bananas, alcohol, or ibuprofen for the 24 h prior to, and during, sample collection. Otherwise, meals were provided by the kitchen staff at CFS Alert and included a variety of options for the participants. The subjects were required to remain awake from 0900 h until 2300 h, after which they could sleep but were awakened in the 15 min prior to each sample. They were released on Sunday morning after the 0900 h sample.

2.3. Saliva collection and melatonin analysis

Saliva was collected from the participants every 2 h using cotton Salivette® collection devices and subsequently frozen at −20 °C. Melatonin content was analyzed by a contract laboratory using RIA kits from Bühlmann Laboratories AG (Schönenbuch, Switzerland). The reported intra- and inter-assay CVs for the kits were 7.9% and 9.8%, respectively, and the limit of detection (analytical sensitivity) was 0.2 pg/ml. The assays were run such that all samples from a given subject were analyzed in duplicate on the same RIA plate. Dim light melatonin onset (DLMO) and offset (MelOFF) were calculated by linear interpolation of the samples that straddled the prescribed threshold of salivary melatonin concentration, 2.7 pg/ml. The duration of melatonin production (DLMO to MelOFF) was calculated for all but two subjects in each trial, as MelOFF could not be calculated for these four subjects due to melatonin production late into the morning (after 0900 h; see Supplementary information). Melatonin rise and fall were calculated at 50% of the amplitude (peak minus trough) after fitting a 6th order polynomial to the data. However, the melatonin profiles of six subjects in January and three subjects in June could not be fitted accurately due to irregularities in their melatonin profiles (e.g. minimal production, spurious peaks, or significantly elevated baseline melatonin). The average fit across the remaining subjects (8 in January, 9 in June) was good (average $r^2 = 0.90 \pm 0.08$ SD).

2.4. Calculation of daily sleep quantity and quality

The quantity of total daily sleep was calculated from the wrist actigraph worn by the subjects. Data was collected in 'zero crossing mode' using one-minute epochs. The start and end of sleep periods were determined by analysis of the actigraphy data, using the manufacturer's software (ActionW version 2.6, Ambulatory Monitoring Inc., Amherst, NY, USA), augmented by sleep log data which described periods of low activity such as reading or watching TV prior to bedtime which might otherwise be mistaken for sleep. Sleep was scored using the Cole–Kripke algorithm [13]. Total sleep time, sleep efficiency, wake after sleep onset (WASO), and the number of sleep episodes were analyzed and compared between the two trials. Total sleep time is defined as the duration of time spent asleep, WASO is defined as the total time spent awake after initial sleep onset and before final awakening, sleep efficiency is defined as the percentage of time spent asleep between sleep onset and final awakening, and the number of sleep episodes is defined as the total number of continuous sleep periods between sleep onset and final awakening. The validity of these measures has been assessed by de Souza and colleagues [14], and a detailed review of the validity and role of actigraphy in sleep and circadian rhythm research has been published by Ancoli-Israel and colleagues [12].

2.5. Measurement and calculation of light exposure

Light exposure data was collected by a photocell on each subject's actigraph and was averaged hourly over the week before the sampling day, and the average and maximum light exposure (lux) determined for each individual. Inspection of the data suggested that for those subjects who had completely different routines on weekends vs. weekdays, the routine they kept for the last two days prior to saliva collection was generally reflected in the results with regard to circadian phase. As the relationship between light and circadian phase shifting is non-linear, we performed a log transformation of the data and then averaged the data 6-hourly from 0700 h for the last two days before sampling (07–13, 13–19, 19–01, 01–07 h). Prior to performing the log transformation, we adjusted the raw light exposure data such that the minimum light intensity was 1 lx. This was done so that the transformed data was always greater than zero, which permitted meaningful averaging of the transformed data.

2.6. Calculation of the melatonin rhythm characteristics

To compare the mean total melatonin production between the January and June photoperiods at CFS Alert, the melatonin profile for each individual was integrated using the trapezoidal rule. Both the total melatonin production for the entire day and the melatonin production between DLMO and dim light melatonin offset (MelOFF) were calculated. Unfortunately we were unable to calculate MelOFF for subjects 28 and 32 for Data Collection 1 (January), and for subjects 3 and 11 for Data Collection 2 (June), primarily because values had not returned to baseline by the end of the sampling time. Therefore these subjects were not included in the calculation of mean melatonin production between DLMO and MelOFF. For the calculation of total daily melatonin production, subjects that did not produce an appreciable amount of melatonin (i.e., peak salivary melatonin ≤ 1 pg/ml) were excluded (subject 29 for Data Collection 2, subject 10 for Data Collection 1).

To compare the duration of melatonin production, the amount of time between DLMO and MelOFF was calculated, and differences between January and June experiments were statistically assessed (Supplementary information). As mentioned above, we were unable to calculate MelOFF for subjects 28, 29 and 32 for Data Collection 2 (January) and for subjects 3, 10 and 11 for Data Collection 3 (June), therefore these subjects were not included in the calculation. Thus, melatonin timing and duration were also calculated from the 50% rise–fall time and assessed in relation to season as above and in relation to sleep timing, duration and light exposure at different times of day during the last two days before sampling.

2.7. Statistics

Statistical differences were assessed using ANOVA (Statistica, StatSoft Inc., Tulsa, OK) when and where appropriate. Details on the type of ANOVA and the layout of the data used for analysis are provided in the description of the statistical results below. Linear regression (Instat 3) was used to determine any relationships of light exposure, sleep timing and duration with melatonin timing, duration and amplitude.

3. Results

3.1. Total daily sleep

The main difference between the research participants in January and June was the quantity of sleep they obtained each night, calculated from the actigraphs. In January, the subjects experienced on average 419 ± 38 (SD) min of sleep each night. In June, the subjects slept on average only 369 ± 43 (SD) min of sleep each night. Therefore, subjects in the June data collection obtained approximately 50 min less sleep each day compared to their January counterparts (Fig. 1A). The differences in sleep quantity between January and June were statistically assessed using a Repeated Measures ANOVA with 'month' as a between factor and 'days' as the repeated measure. A significant main effect of 'month' indicated that the mean quantity of sleep was significantly different between January and June [$F(1, 23) = 4.98$, $p = .036$] (Fig. 1A). A significant main effect of 'days' [$F(5, 115) = 3.87$, $p = .003$], followed-up with a simple linear regression analysis of the daily mean sleep minutes ($r = 0.83$, $p = 0.04$), indicated that the subjects received a significantly reduced quantity of sleep as the week progressed (Fig. 1B); however, the 'days' × 'month' interaction was not significant [$F(5, 115) = .38$, $p = .86$], which indicates that this sleep behavior was similar in January and June, as illustrated in Fig. 1B. Post-hoc analysis (Tukey HSD) found that the only the significant differences between individual data points were between Sunday (January) and Wednesday (June; $p = 0.007$), and Sunday (January) and Friday (June; $p = 0.001$).

3.2. Melatonin production during January and June in relation to sleep

Total daily melatonin production was calculated first by integrating the entire melatonin profile of each subject, and then by only integrating the melatonin profile between DLMO and MelOFF. The mean total daily melatonin production (±SD) was found to be 162.67 ± 120.55 in January vs. 112.74 ± 67.33 pg/ml in June (Fig. S1A). Mean melatonin production (±SD) between DLMO and MelOFF was found to be 142.34 ± 122.88 pg/ml in January, and 104.77 ± 74.09 pg/ml in June (Fig. S1B). Despite the notably higher production of melatonin during the winter months in CFS Alert, these differences were not statistically significant regardless of the method used to calculate total melatonin production. The duration of melatonin production between January and June was also not statistically different. In January, the mean duration of melatonin production was found to be $11.78 \text{ h} \pm 1.09$ h, and in June the mean duration of melatonin production was found to be $10.39 \text{ h} \pm 0.76$ h (mean ± SEM; Fig. S1C). A one-way ANOVA was used to test for statistical significance (January and June) [$F(1, 17) = 0.837$, $p = 0.37$].

3.3. Comparison of melatonin timing and amplitude with evening light exposure and sleep–wake times in June

As for total melatonin production, the amplitude did not differ between seasons (see Supplementary information, Fig. S1A and S1B).

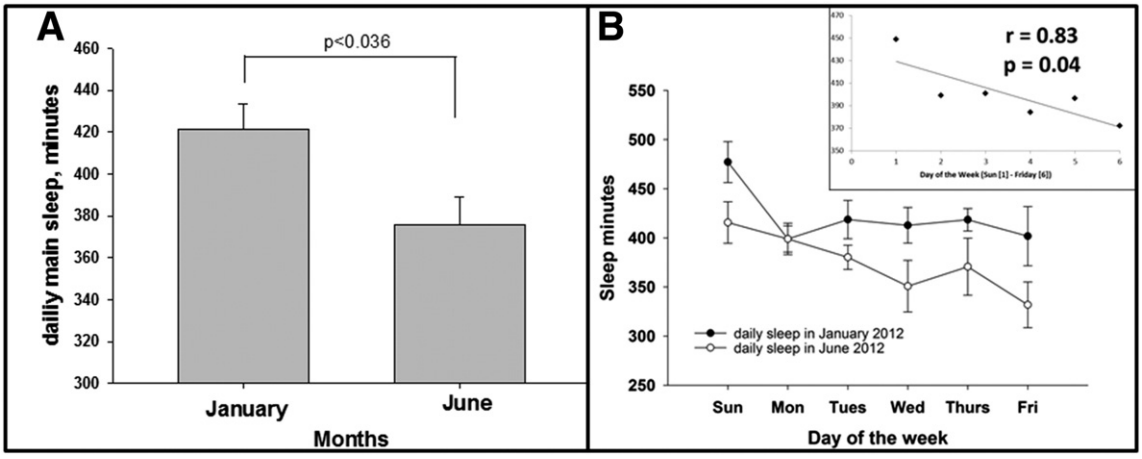

Fig. 1. A. Main effect of 'month' for daily main sleep period is plotted for each of the January and June 2012 data collections at CFS Alert. Both values are mean ± s.e.m. B. The mean sleep minutes of the daily main sleep period are plotted over days for each of the January and June 2012 data collections at CFS Alert. Solid circles are January subjects and open circles are June subjects. All values are means ± s.e.m.

Since the quantity of melatonin produced did not statistically correlate to quantity of sleep (see Supplementary information, Fig. S1E), and therefore does not explain the sleep difficulties experienced in June, we looked at the relationships between melatonin DLMO and rise/fall timing with sleep timing in both June and January. There were no significant relationships in January (see Fig. 2). In June statistically significant correlations were found between melatonin 50% rise times with sleep end (arise) times, and between melatonin 50% fall times with sleep start and end (arise) timing (Fig. 3). The graphs in Fig. 4A and B also highlight the much delayed melatonin onset that occurred in June.

Notably, statistically significant correlations were also found between melatonin rise/fall times and evening light exposure (Fig. 4A and B) from the June, but not the January data, which suggests that evening light exposure may be contributing to delaying melatonin production onset.

3.4. Mistimed sleep relative to the circadian system in the Arctic summer affects sleep quality

The June study participants were categorized based on sleep time and DLMO time. Sleep onset normally occurs 1–2 h after melatonin onset [15,16]. Those participants that had a mean sleep start time (bedtime plus sleep latency) that came before their DLMO were placed into one group, subsequently called 'mistimed sleepers'. The participants that had a mean sleep start time that came after their DLMO were placed into a separate group, subsequently called 'appropriately-timed sleepers'. Only one subject, S6, was omitted from the analysis because their work schedule was highly variable, and often had to be on-duty at night. Sleep quantity and quality parameters were subsequently analyzed for these two groups in June. The mean quantity of sleep (±SD) obtained each night for the 'mistimed sleepers' was 360.48 ± 71.94 min (Sunday night to Friday night). Mean sleep efficiency was 92.54 ± 4.64%, mean wake-after-sleep-onset (WASO) was 29.90 ± 21.00 min, and the mean number of sleep episodes was 12.69 ± 5.93. For the 'appropriately-timed sleepers', the mean quantity of sleep obtained each night was 395.44 ± 76.95 min (Sunday night to Friday night), mean sleep efficiency was 95.88 ± 5.09%, mean WASO was 17.31 ± 22.48 min, and the mean number of sleep episodes was 6.33 ± 3.61. A Between-Groups Repeated Measures ANOVA was used to compare the sleep data with 'group' as the between subjects factor and 'days' as the repeated measure. This revealed that the groups were statistically independent for all of the aforementioned parameters (Fig. 5). There was no statistical effect of 'days', nor was there an interaction between the factors. In summary, the mistimed sleepers received significantly less sleep each night [$F(1, 54) = 4.85$, $p = 0.032$], and had a significantly lower sleep efficiency [$F(1, 53) = 6.55$, $p = 0.013$], a significantly higher WASO [$F(1, 53) = 4.53$, $p = 0.038$], and a significantly greater number of sleep episodes [$F(1, 53) = 18.84$, $p = 0.00006$].

4. Discussion

The comparison of sleep data between January (24 h darkness) and June (24 h sunlight) from participants at CFS Alert indicates that there is a significant reduction of sleep among military residents in the high arctic during the summer months, amounting to 50 min on average each day. For both January and June participants, the quantity of sleep obtained each night as the week progressed from Sunday to Friday decreased. A reduced quantity of daily sleep during the work-week, compared to the weekend, is common among working adults in America according to data from the 2008 National Sleep Foundation's (NSF) Sleep in America poll [17,18]. For January participants, the mean quantity of sleep for any given night remained above 400 min (6 h 40 min); however, for June, mean quantity of sleep decreased steadily each night to only 330 min (5.5 h) on the Friday night before saliva collection in June (Fig. 1). Such short sleep duration is not considered sufficient to maintain optimal cognitive performance from day to day. We suspect that the reduced duration of sleep obtained each night in June highlights the prevalence of sleep difficulties during the arctic summer, which is

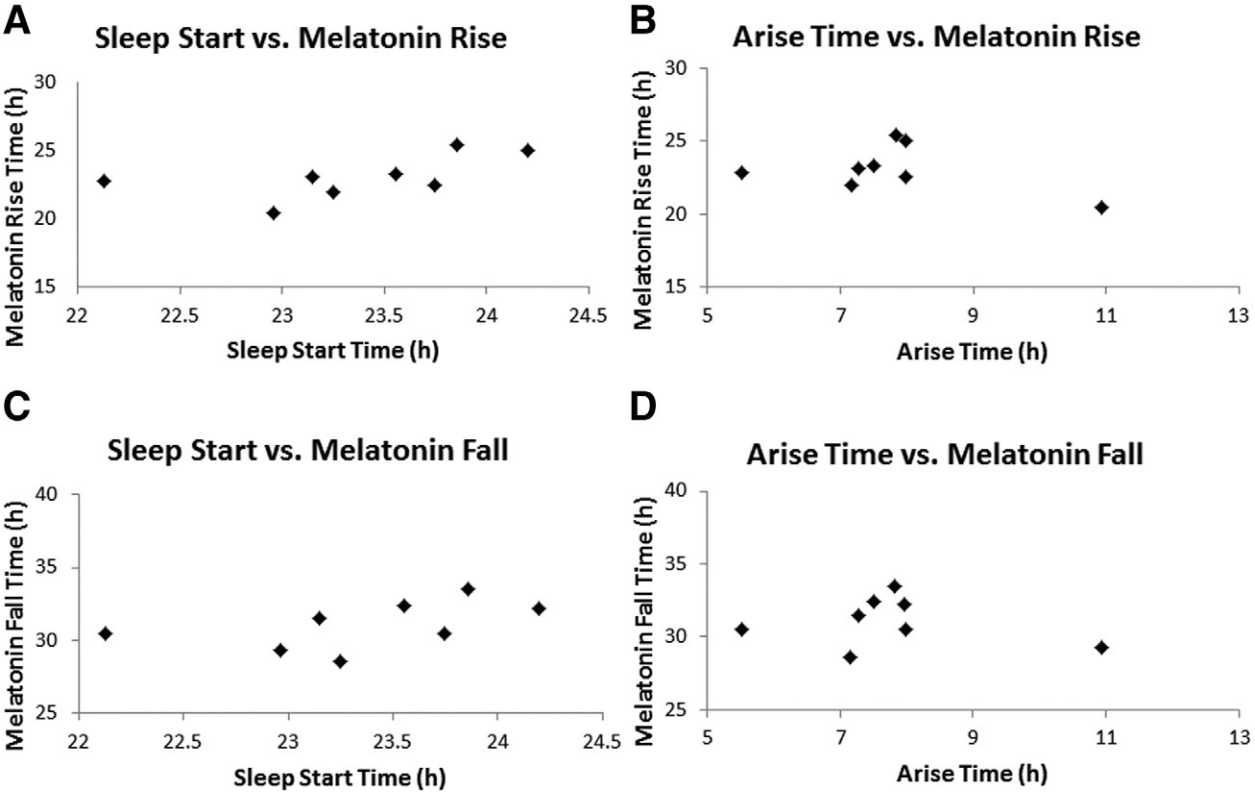

Fig. 2. Correlation between melatonin rise/fall with sleep start/end times in January.

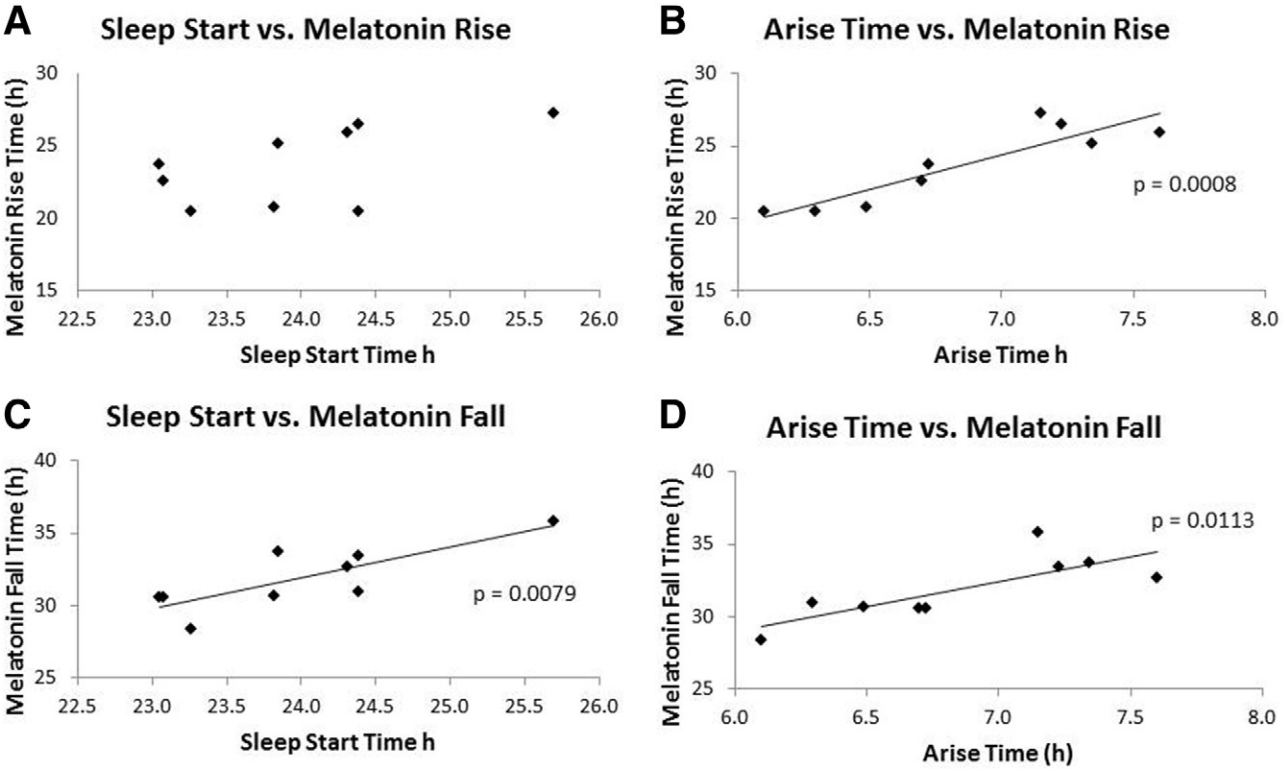

Fig. 3. Correlation between melatonin rise/fall with sleep start/end times in June.

only compounded by work requirements and social behaviors late in the work week (Wednesday–Friday).

Our initial hypothesis – that a changing duration of melatonin in extreme photoperiods would influence sleep – was not supported, since neither the duration nor the amplitude of the melatonin rhythm differed significantly between seasons. This is consistent with some previous Antarctic data [19] and with studies artificially extending the melatonin profile together with an extended sleep opportunity. In the latter case sleep was differently distributed during the 16 h sleep opportunity without an increase in total sleep time [20]. However the reduction of melatonin production during the summer months, and a correlation analysis between melatonin production and sleep duration indicates that there is a trend for those who produce less melatonin to sleep less, and those who produce more melatonin to sleep more, but this trend was not statistically significant. The relationship between melatonin production, sleep and photoperiods has already been established by Wehr, in controlled conditions. He exposed eight healthy volunteers long-term to artificial 'summer' and 'winter' photoperiods and found that the durations of melatonin secretion and sleep-phase were both longer after exposure to the short 'winter' photoperiod [21]. Wehr demonstrated that the onset and/or offset of melatonin production coincides with the nocturnal period of sleepiness [22], which leads us to consider that the reduced quantity of melatonin production in June may be partially responsible for the reduction in total sleep time. There is also good evidence that natural 'long' sleepers have a longer biological night as evidenced by melatonin duration [23]. However, more numbers are required before any firm conclusions can be drawn from our observations.

It is clear from our data that a major factor influencing sleep quantity and quality during Arctic residence is the timing of sleep relative to

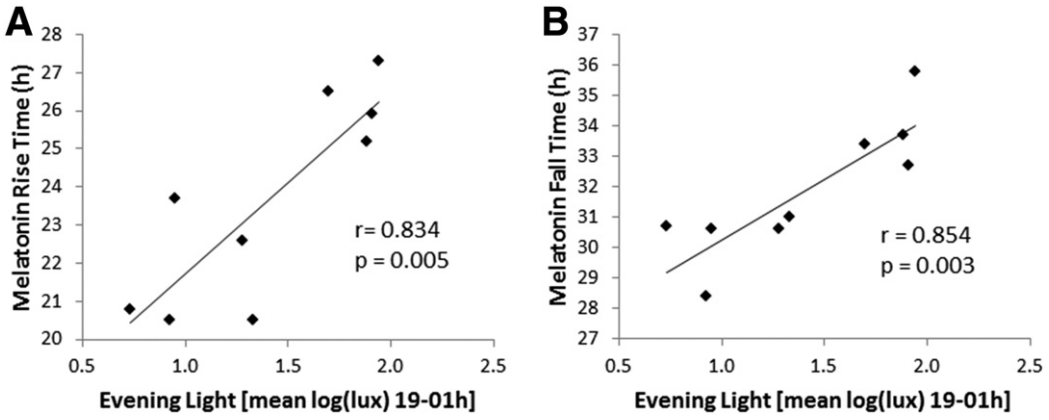

Fig. 4. Correlation between melatonin rise/fall and evening light exposure in June. A. Melatonin rise time is correlated to light exposure in the evening (1900 h to 0100 h). B. Melatonin fall time is correlated to light exposure in the evening (1900 h to 0100 h).

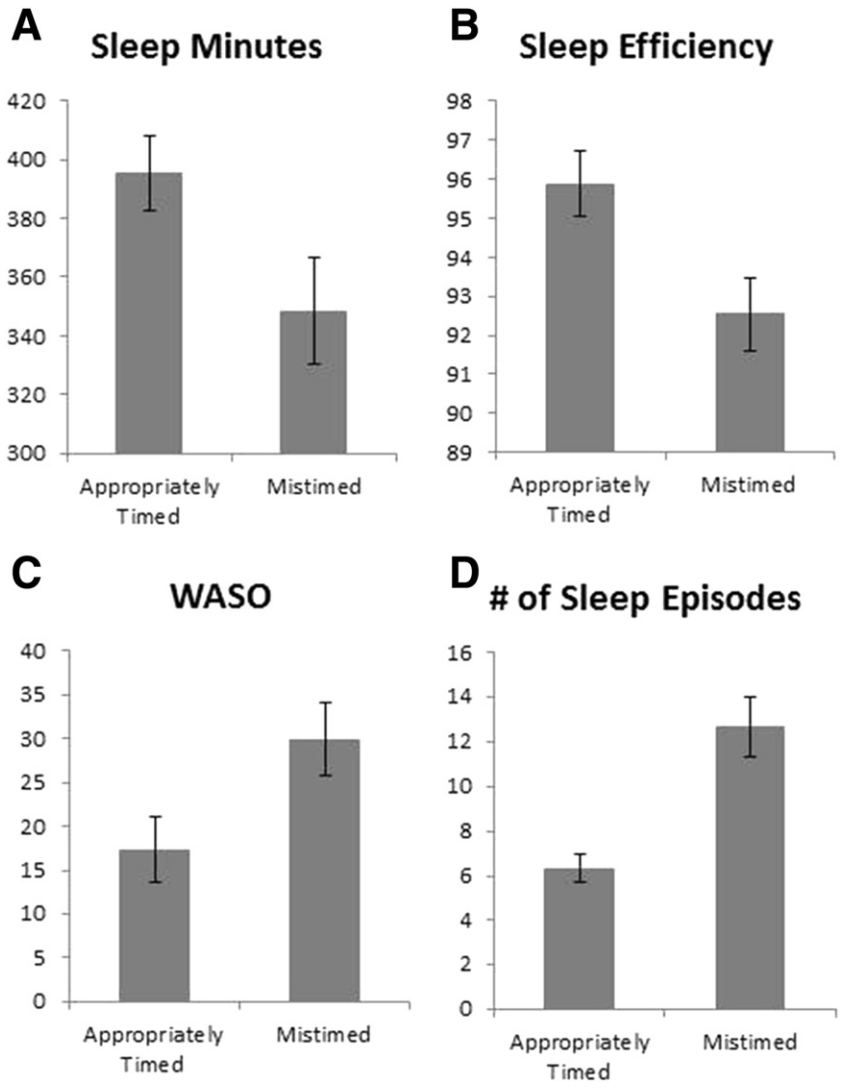

Fig. 5. Analysis of sleep quantity and quality for individuals that went bed before DLMO (mistimed sleepers), and those that went to bed after their DLMO (appropriately timed sleepers). The two populations are statistically independent on all measures shown above.

internal circadian phase. This is likely true regardless of the season, though we only found summer residents to be at risk of mistimed sleep relative to internal circadian phase. Due to the absence of bright sunlight in the winter and the continuity of bright sunlight in the summer, the movement of circadian phase from inappropriately timed light exposure on the weekend may have been less substantial in the winter relative to the summer. As Fig. 2 shows, there are statistically significant correlations between sleep start and end times (i.e. sleep onset and arise time), and melatonin rise and fall times. Perhaps not surprisingly, melatonin rise and fall times are also significantly correlated to evening light exposure in June, which suggests that evening light exposure may be contributing to the delayed melatonin production observed in many of the participants in June (Figs. 2 and 3). The relationship between evening light exposure and melatonin rise/fall times may also be due to the tendency of individuals with a late melatonin rhythm to go to bed later and thus expose themselves to high amounts of light in the evening [16], and this exposure may contribute to further delaying melatonin production in these individuals.

Sleep onset normally occurs 1–2 h after the DLMO but in many cases sleep onset preceded the DLMO. Analyzing the data with individuals categorized according to the synchronization of their sleep time and DLMO revealed that there was a prevalence of individuals with a sleep schedule that was out of alignment with their melatonin rhythm. The 'mistimed sleepers' not only obtained less sleep each night, but also woke up more frequently during the night and were awake for more time during the night after they had fallen asleep. Therefore, both the quantity and quality of sleep obtained each night by the 'mistimed sleepers' were lower compared to those individuals that maintained a sleep schedule that was aligned with their endogenous melatonin rhythm. Considering this differentiation of the two groups in June, the statistically significant correlations between melatonin rise/fall times and evening light suggests that exposure to light late in the day on the delay portion of the human PRC is likely to be responsible for the delayed melatonin production by the mistimed participants in June. In other words, the increased light exposure in the evening promoted a later melatonin rise time and a later bedtime. However, the subjects in June did not carry out any compensatory behaviors to prevent drifting of the circadian rhythm. All of our subjects worked indoors and were not exposed to the bright outdoor sunlight during mornings that may have otherwise anchored their circadian rhythms. Arise time was also correlated to the rise and fall of melatonin; however our participants were restricted to set work and meal schedules which did not allow them to wake in accordance with their body's rhythms. This may have also contributed to the shorter sleep durations for this group, as their later melatonin rise time would naturally lend itself to a wake time later than their schedule allows.

This study provides an analysis of the sleeping patterns and melatonin rhythms of military personnel stationed at an extreme northern latitude (82.5° N). It is an observational study, and due to the small sample size of each trial, there is limited generalizability of the results. There are also confounding factors that we could not take into account, notably the different individuals in each season. However, while there is some inconsistency in the observations at high latitudes, several studies have shown similar results with regard to seasonality in sleep duration. For example Nixon and colleagues have also shown longer sleep during the winter compared with summer in Auckland, New Zealand [24], and Lehnkering and Siegmund showed longer sleep during the autumn compared with the spring in Berlin, Germany [25]. Thorleifsdottir and colleagues found that Icelanders sleep longer during the winter as well, but showed this tendency to be age-dependent [26]. On the other hand a large epidemiological study found no differences in sleep duration across the four seasons, but the results were based solely on questionnaires [27]. When comparing sleep-logs from University students in Norway, Friborg found there to be a delay in sleep timing during the winter compared to summer, but no difference in sleep duration across the seasons [28,29]. Our primary concern from the collection and analysis of the data presented here is that individuals were sleeping substantially less in the summer in these particular conditions. Such lack of sleep in the summer is likely to impact cognitive performance, and thus military operational readiness.

One may question if confounders varied with the results, and although a thorough check is not possible, we do not believe that were any variables that may have affected the melatonin rhythms or sleep of our participants: physical activity was reported and observed to be similar between the seasons; indoor lighting levels did not change between the seasons; work schedules remained consistent; and the participants were always provided with the opportunity to darken their bedrooms before going to bed.

In conclusion, the presence of continuous sunlight during the arctic summer leads to a reduction in the quantity of sleep obtained by residents of CFS Alert in June, relative to January residents. There is a prevalence of individuals with misaligned sleep and melatonin rhythms in June who obtain sleep that is of a reduced quantity and quality. Regardless of the cause, reduced sleep quantity for long periods of time is troubling because of its direct effect on cognitive performance [30–32]. It is well known that a chronic sleep debt adversely affects cognitive performance, and therefore the promotion of additional sleep during the summer months in the high arctic will be the focus of subsequent trials. Given the results from this study, appropriate countermeasures for sleep promotion during summer in the high arctic may include the prescription of special sunglasses that filter out blue and green light wavelengths, which would be worn 2–3 h before an appropriate bedtime to create “circadian darkness” and thus facilitate appropriately timed melatonin production and indeed the timing of the circadian in general. The prescription of exogenous melatonin prior to bedtime may also help with facilitating sleep by simulating ‘physiologic night’.

Support

Canada DND Research Project 04KD07 was awarded to MAP.

Disclosure

The authors have nothing to disclose.

Conflicts of interest

MAP, RJL, and AH have no conflicts of interest to report. JA is a stockholder and consultant for Stockgrand Ltd and Surrey Assays Ltd. This manuscript is not related to these relationships.

Author contributorship

MAP and JA designed the study. AH and MAP collected the data. All authors contributed to data analysis, interpretation of results, and manuscript preparation.

Acknowledgments

This research was supported by the Canadian Department of National Defence (Research Project 04KD07).

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.physbeh.2015.01.021.

References

[1] S. Pallesen, I.H. Nordhus, G.H. Nielsen, O.E. Havik, G. Kvale, B.H. Johnsen, et al., Prevalence of insomnia in the adult Norwegian population, Sleep 24 (2001) 771–779.
[2] K. Obayashi, K. Saeki, J. Iwamoto, N. Okamoto, K. Tomioka, S. Nezu, et al., Effect of exposure to evening light on sleep initiation in the elderly: a longitudinal analysis for repeated measurements in home settings, Chronobiol. Int. 31 (2014) 461–467.
[3] J. Arendt, Biological rhythms during residence in polar regions, Chronobiol. Int. 29 (2012) 379–394.
[4] A. Kauppila, A. Kivela, A. Pakerinen, O. Vakkuri, Inverse seasonal relationship between melatonin and ovarian activity in humans in a region with a strong seasonal contrast in luminosity, J. Clin. Endocrinol. Metab. 65 (1987, Nov) 823–828.
[5] L. Makkison, J. Arendt, Melatonin secretion on two different Antarctic bases, J. Interdiscip. Cycle Res. 22 (1991) 149–150.
[6] T. Paakonen, J. Leppaluoto, T.M. Makinen, H. Rintamaki, A. Ruokonen, J. Hassi, Seasonal levels of melatonin, thyroid hormones, mood, and cognition near the Arctic Circle, Aviat. Space Environ. Med. 79 (Jul 2008) 695–699.
[7] N. Rosenthal, D. Sack, F. Jacobsen, S. James, B. Parry, J. Arendt, et al., Melatonin in seasonal affective disorder and phototherapy, J. Neural Transm. Suppl. 21 (1985) 257–267.
[8] M.A. Paul, G.W. Gray, H.R. Lieberman, R.J. Love, J.C. Miller, M. Trouborst, et al., Phase advance with separate and combined melatonin and light treatment, Psychopharmacology (Berl.) 214 (2011) 515–523.
[9] M.A. Paul, J.C. Miller, R.J. Love, H.R. Lieberman, S. Blazeski, J. Arendt, Timing light treatment for eastward and westward travel preparation, Chronobiol. Int. 26 (2009) 867–890.
[10] M.A. Paul, J.C. Miller, G.W. Gray, R.J. Love, H.R. Lieberman, J. Arendt, Melatonin treatment for eastward and westward travel preparation, Psychopharmacology (Berl.) 208 (2010) 377–387.
[11] M.A. Paul, R.J. Love, A.M. Hawton, K. Brett, D.R. McCreary, J. Arendt, Sleep deficits in the High Arctic summer in relation to light exposure and behaviour: use of melatonin as a countermeasure, Sleep Med. (2014). http://dx.doi.org/10.1016/j.sleep.2014.12.012 (Accepted Dec 2014).
[12] S. Ancoli-Israel, R. Cole, C. Alessi, M. Chambers, W. Moorcroft, C.P. Pollak, The role of actigraphy in the study of sleep and circadian rhythms, Sleep 26 (2003) 342–392.
[13] R.J. Cole, D.F. Kripke, W. Gruen, D.J. Mullaney, J. Gillin, Automatic sleep/wake identification from wrist activity, Sleep 15 (1992) 461–469.
[14] L. de Souza, A.A. Benedito-Silva, M.L.N. Pires, D. Poyares, S. Tufik, H.M. Calil, Further validation of actigraphy for sleep studies, Sleep 26 (2003) 81–85.
[15] H.J. Burgess, C.I. Eastman, The dim light melatonin onset following fixed and free sleep schedules, J. Sleep Res. 14 (2005) 229–237.
[16] K.P. Wright Jr., C. Gronfier, J.F. Duffy, C.A. Czeisler, Intrinsic period and light intensity determine the phase relationship between melatonin and sleep in humans, J. Biol. Rhythms 20 (2005) 168–177.
[17] T.J. Balkin, G. Belenky, C. Drake, R. Rosa, M. Rosekind, Sleep in America poll: summary of findings, Natl. Sleep Found. 2008 (2008) 45.
[18] L.M. Swanson, J.T. Arnedt, M.R. Rosekind, G. Belenky, T.J. Balkin, C. Drake, Sleep disorders and work performance: findings from the 2008 National Sleep Foundation Sleep in America poll, J. Sleep Res. 20 (2011) 487–494.
[19] J. Broadway, J. Arendt, S. Folkard, Bright light phase shifts the human melatonin rhythm during the Antarctic winter, Neurosci. Lett. 79 (1987) 185–189.
[20] S.M.W. Rajaratnam, B. Middleton, B.M. Stone, J. Arendt, D.J. Dijk, Melatonin advances the circadian timing of EEG sleep and directly facilitates sleep without altering its duration in extended sleep opportunities in humans, J. Physiol. 339–51 (2004).
[21] T.A. Wehr, The durations of human melatonin secretion and sleep respond to changes in daylength (photoperiod), J. Clin. Endocrinl. Metab. 73 (1991) 1276–1280.
[22] T.A. Wehr, D. Aeschbach, W.C. Duncan Jr., Evidence for a biological dawn and dusk in the human circadian timing system, J. Physiol. 535 (2001) 937–951.
[23] D. Aeschbach, L. Sher, T.T. Postolache, J.R. Matthews, M.A. Jackson, T.A. Wehr, A longer biological night in long sleepers than in short sleepers, J. Clin. Endocrinol. Metab. 88 (2003) 26–30.
[24] G.M. Nixon, J.M.D. Thompson, D.Y. Han, D.M. Becroft, P.M. Clark, E. Robinson, et al., Short sleep duration in middle childhood: risk factors and consequences, Sleep 31 (2008) 71–78.

[25] H. Lehnkering, R. Siegmund, Influence of chronotype, season, and sex of subject on sleep behavior of young adults, Chronobiol. Int. 24 (2007) 875–888.
[26] B. Thorleifsdottir, J.K. Bjornsson, B. Benediktsdottir, T. Gislason, H. Kristbjarnarson, Sleep and sleep habits from childhood to young adulthood over a 10-year period, J. Psychosom. Res. 53 (2002) 529–537.
[27] M.T. Johnsen, R. Wynn, T. Bratlid, Is there a negative impact of winter on mental distress and sleeping problems in the subarctic: the Tromso Study, BMC Psychiatry 12 (2012).
[28] O. Friborg, B. Bjorvatn, B. Amponsah, S. Pallesen, Associations between seasonal variations in day length (photoperiod), sleep timing, sleep quality and mood: a comparison between Ghana (5 degrees) and Norway (69 degrees), J. Sleep Res. 21 (2012) 176–184.
[29] O. Friborg, J.H. Rosenvinge, R. Wynn, M. Gradisar, Sleep timing, chronotype, mood, and behavior at an Arctic latitude (69 degrees N), Sleep Med. 15 (2014) 798–807.
[30] D.J. Dijk, J.F. Duffy, C.A. Czeisler, Circadian and sleep/wake dependent aspects of subjective alertness and cognitive performance, J. Sleep Res. 1 (1992) 112–117.
[31] D.F. Dinges, F. Pack, K. Williams, K.A. Gillen, J.W. Powell, G.E. Ott, et al., Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night, Sleep 20 (1997) 267–277.
[32] N. Goel, H. Rao, J.S. Durmer, D.F. Dinges, Neurocognitive consequences of sleep deprivation, Semin. Neurol. 29 (2009) 320–339.